Stomach and duodenum

Objectives:

- The surgical anatomy of stomach and duodenum, their blood supply, nerve supply & lymphatics.
- The surgical physiology of gastric motility and secretions.
- Special forms of peptic ulceration.
- Management of uncomplicated peptic ulcer disease.
- Complications of peptic ulceration requiring operative intervention including:
    - Perforation (perforated ulcers).
    - Acute hemorrhage.
    - Pyloric stenosis.
- Benign Gastric neoplasms.

Resources:

- Davidson's.
- Slides
- Surgical recall.
- Raslan's notes.
- Current diagnosis and treatment (colored in blue)
- Team 434

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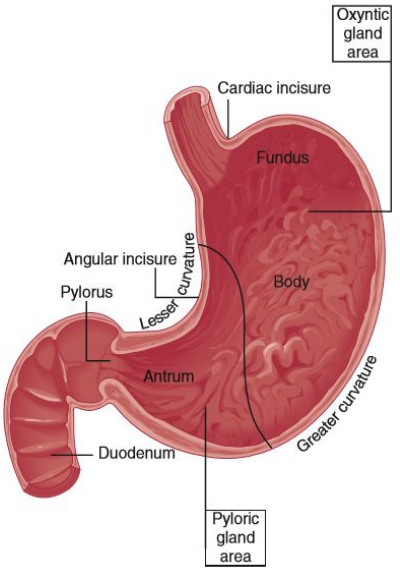
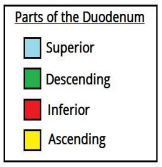
Basic review(anatomy):

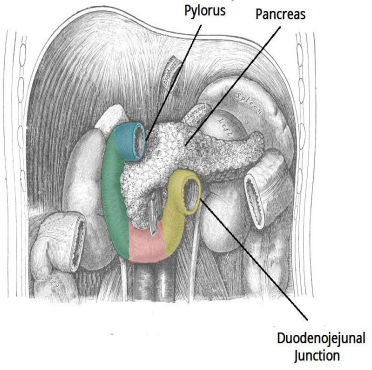
Doctor said:Stomach & Duodenum anatomy (including its blood supply & their origins) and physiology are included in the examination and you need to cover it(check the MCQ's in the last page)

Stomach subdivided into 5 parts (each part has secretory and nonsecretory function)	
Part	Notes
Cardia	Site for Barrett's disease
Fundus	Secretory: parietal cell and Neurogenic: hunger feeling
Body	Secretory: parietal cell
Antrum	Site for surgical ulcer treatment: by cutdown the acid secretion (site for gastrin) Even if the ulcer in the duodenum.
Pylorus	Site for Dumping Syndrome (food goes immediately to SI)



Duodenum parts : 25 cm long and subdivided into 4 parts			
Part	**Name**	**Level**	**Common site for**
First	Upper	1st LV	Ulcer
Second	Vertical	2nd LV	**Diverticulum**
Third	Horizontal	3rd LV	**Superior Mesenteric Artery Syndrome**
Fourth	Ascending	3rd LV	-

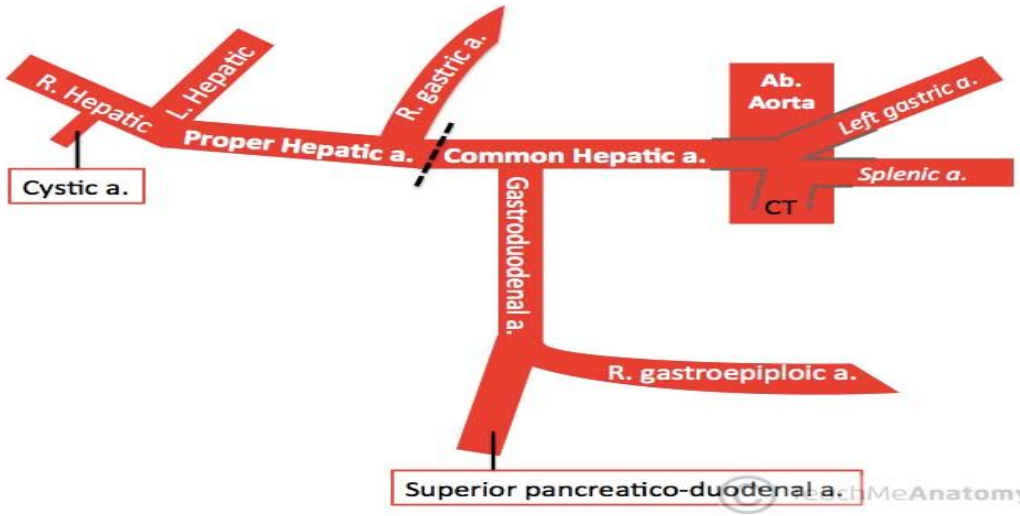




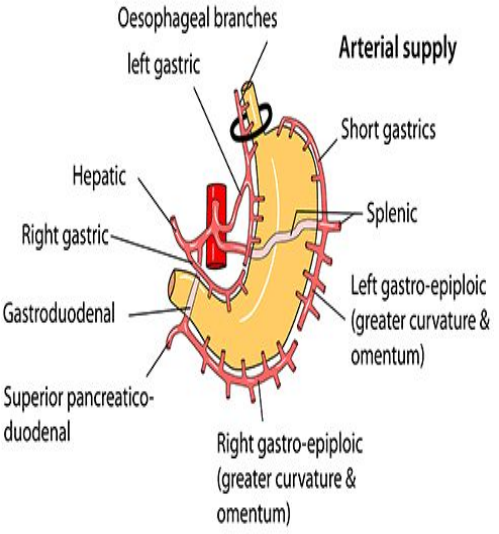
The 1st part of Duodenum: (Relations)

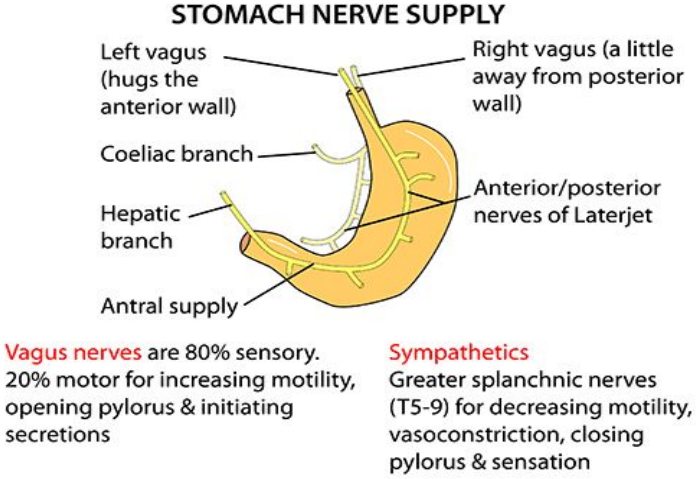
- Anteriorly:The quadrate lobe of the liver and the gallbladder .
- Posteriorly: The lesser sac (first inch only), the gastroduodenal artery (that's why Posterior ulcer bleed), the bile duct and portal vein, and the inferior vena.
- Superiorly:The entrance into the lesser sac (the epiploic foramen)
- Inferiorly: The head of the pancreas

Basic review(anatomy):



Blood supplies		
Artery	**Branch**	**Supply**
Celiac trunk	Left gastric	Esophageal branches
Splenic a.	Left gastroepiploic	Supply the greater curvature of the stomach. Anastomosis with the right gastroepiploic artery.
	Short gastric arteries	5-7 small branches supplying the fundus of the stomach.
	Pancreatic branches	Supply the body and tail of pancreas
Common hepatic a. Proper hepatic a.	Right gastric	Supplies the pylorus and lesser curvature of the stomach
Common hepatic a. Gastroduodenal a.	Right gastroepiploic	Supplies the greater curvature of the stomach. Found between the layer of greater omentum, which is also supplies.
Gastroduodenal a.	Superiore pancreaticoduodenal	Divides into an anterior and posterior branch, which supplies the head of the pancreas.





Basic review(physiology of the stomach): Source of this part is Davidson's

Gastric Motility:

Food is passed from the oesophagus into the stomach, where it is stored, ground and partially digested. As food enters the stomach:

1. the muscles in the stomach walls relax and intragastric pressure rises only slightly. This effect is known as **receptive relaxation**, and is mediated by the vagus nerve.
2. It is followed by muscular contractions that increase in amplitude and frequency, starting in the fundus and moving down towards the body and antrum. In the antrum, the main role is the grinding of food and propulsion of small amounts (now called chyme) into the duodenum when the pyloric sphincter relaxes.

Gastric emptying is controlled by two mechanisms: **hormonal feedback** and **a neural reflex called the enterogastric reflex**.

1. In the former, fat in the chyme is the main stimulus for the production of a number of hormones, the most powerful being cholecystokinin, which exerts a negative feedback effect on the stomach, decreasing its motility.
2. The enterogastric reflex is initiated in the duodenal wall, and this further slows stomach emptying and secretion.

Gastric Secretions:

Classically, gastric secretion has been divided into three phases:

1. **Cephalic (neural) phase.** Signals arise in the central cortex or appetite centres, triggered by the sight, smell, taste and thought of food, and travel down the vagus nerves to the stomach.
2. **Gastric phase**. Food (in particular protein digestion products) causes the release of acid, this release controlled by a negative feedback mechanism dependent upon the pH of the stomach. The gastric phase accounts for the greatest part of daily secretion, approximately 1.5 litres
3. **Intestinal phase.** The presence of food in the duodenum triggers the release of a number of hormones, including duodenal gastrin. These exert a positive feedback effect on the stomach, causing a small increase in gastric secretion.

- **Mucus** is produced by all regions of the stomach. It is composed mainly of glycoproteins, water and electrolytes, and serves two important functions. It acts as a lubricant, and it protects the surface of the stomach against the powerful digestive properties of acid and pepsin. Bicarbonate ions are secreted into the mucus gel layer and this creates a protective buffer zone against the effects of the low pH secretions. Alkaline mucus is produced in the duodenum and small intestine, where it has a similar function of mucosal protection.

- **The parietal cells in the stomach** are responsible for the production of acid. Acid secretion by these cells is stimulated by two main factors: acetylcholine, released by the vagus nerve, gastrin from the antrum, and direct contact. Acetylcholine and gastrin act on neuroendocrine cells located close to the parietal cells. On stimulation, these cells release histamine, which has a paracrine action on the parietal cell, stimulating acid production and secretion. Parietal cells secrete acid via an active transport mechanism, the proton pump. Somatostatin, gastric inhibitory peptide and vasoactive intestinal peptide inhibit acid secretion.
- **Pepsin** is a proteolytic enzyme produced in its precursor form, pepsinogen, by the peptic cells found in the body and fundus of the stomach. Pepsinogen production is stimulated by acetylcholine from the vagus nerve. The precursor is then converted to its active form, pepsin, by the acid contents of the stomach.
- **Intrinsic factor** is also produced by the parietal cells. It is a glycoprotein that binds to vitamin B_{12} present in the diet and carries it to the terminal ileum. Here specific receptors for intrinsic factor exist and the complex is taken up by the mucosa. Intrinsic factor is broken down and vitamin B_{12} is then absorbed into the bloodstream.

Peptic ulcer

زمان اول ما اكتشفوا الالسر كان على بالهم ان السبب الوحيد له هو زياده الاسديتي فقالوا يله خن نسمي الالسر الي يجي بالجي اي (بيبتك ألسر) بيبتك يعني اسيد!_بس حديثا اكتشفوا انه مو بس الاسيديتي العاليه تسبب ألسر بل حتى القاعديه العاليه ممكن تسببه!!بس للحين ماز الوا يستخدمون التسميه القديمه(peptic ulcer)الي ممكن تكونmisleading في بعض الاحيان!!!

General Considerations:

- **Sites of Peptic ulcer:** (ulceration in any acid-producing organ)
  - Esophagus
  - Stomach
  - Duodenum
  - Jejunum (following a gastrojejunostomy)
  - Ileum (in relation to ectopic gastric mucosa in Meckel's diverticulum)

  (Most common cause of epigastric pain related to the stomach and the duodenum)
- **Men** are affected **three times** as often as women.
- Duodenal ulcers are **ten times more** common than gastric ulcers in **young** patients.
- In the **older** age groups the **frequency is about equal**

Clinical presentation:

Pain - epigastrium (well localized)	**Bleeding** - permouth or perrectum	**Vomiting**- due to obstruction

Duodenal Ulcer vs Gastric Ulcer:

	Duodenal Ulcer	Gastric Ulcer
Clinical features	1. Well localized epigastric pain (mid-day, noon and night) 2. Pain when hungry , and is relieved by food (increase in weight)	1. Epigastric pain 2. The pain occur during eating (produce more acid) and is relieved by vomiting (decrease in weight) **VERY IMP:**Gastric ulcer may develop into malignancy much more often than duodenal ulcers *Always biopsy a gastric ulcer – some will be malignant *Elderly comes complaining of epigastric pain which is worsen by food → most likely gastric ulcer. U should do EGD to know the site of ulcer ,if it in the stomach u should take biopsy to exclude malignancy.
Age	Common in young and middle-aged males	Common in 40-60 years male
Site	95% occur in duodenum bulb (2 cm),(the 1st part of duodenum)	95% occur along lesser curve, (where is the incisura angularis)[1]. See the 1st page for a pic :)
Note	- Normal or increased acid secretion - 90% caused by helicobacter pylori[2] very imp ,very common exam question - Typical presentation : young male complaining of epigastric pain - especially while fasting - which is relieved by eating. it's most likely duodenal ulcer caused by H.pylori ,give him drug to eradicate h.pylori ,no need to do scopy	**Types**: it's not imp to know the types bc all of them treated with the same treatment - Type1: in incisura angularis & normal acid - Type 2: ulcers are located close to the pylorus and occur in association with duodenal ulcers& high acid (most common) - Type3: in antrum, due to chronic use of NSAIDs - Type4:At the gastroesophageal junction

[1] The most common site of gastric ulcer is incisura angularis, the 2nd most common site is Pylorus.
[2] H.pylori is GNCB aerophilic.

Diagnosis:

- EGD (Esophagogastroduodenoscopy) best maneuver [3] / EGD with biopsy (Biopsy is important in gastric ulcer to exclude malignancy)
  - Gastric analysis:
    - Basal vs maximal (not practical and isn't used nowadays)
  - Gastrin serum level:
    - Severe or refractory (done if zollinger-ellison syndrome is suspected or the treatment was not effective).
  - Contrast meal:
    - Used when either endoscopy is contraindicated or complications of ulcer have occurred
      - Contrast swallow visualizes esophagus only
      - Contrast meal visualizes stomach and esophagus
      - Contrast follow through visualized stomach, esophagus and small bowel

Before doing all the test, you must first treat the patient if you suspect duodenal ulcer for at least 6 weeks, if he didn't recover or the ulcer recurred → scope.

Treatment:

Medical Treatment (80% in 6 weeks)	**Surgical Treatment**
• H2 antagonist (eg.Zantac) - control acid secretion • Proton pump inhibitors PPI (eg.Omeprazole) (strong one)"PPI→block the ACTIVE secretion of hydrogen ions into the stomach so it can't combine with chloride ions in the stomach lumen to form gastric acid" • Antibiotics (eg.Amoxicillin, Clarithro): for H. Pylori eradication	[it has been limited to patients in whom complications have occurred or to block hormonal stimulation] • Vagotomy (block the neuronal stimulation →which blocks hormonal stimulation) • Antrectomy[4] + vagotomy • Subtotal gastrectomy

Complications Of Surgery For Peptic Ulcer

Early Complications: leakage, bleeding, gastric retention (poor gastric emptying), perforation

Late Complications أهم أهم كومبليكيشن لازم تعرفونها الانيميا والدومبينق

1. Recurrent ulcer (marginal ulcer, stomal ulcer ,anastomotic ulcer)
2. Gastrojejunocolic and gastrocolic fistula
3. **Dumping syndrome:** A condition where the ingested food bypasses the stomach too rapidly and enters the small intestine largely undigested. B/c there is no pylorus due to surgery → undigested food will go to small bowel directly, this food has an osmotic potential(it's hyperosmolar) thus it drags fluid,occurs 1-3 hours after a meal.
   - **Early dumping:** It happens when the duodenum, expands too quickly due to the presence of hyperosmolar food from the stomach.leading to hypovolemia (Syncope, loss of consciousness and lethargy) → takes 4 -6 mins.
   - **Late dumping:** is due to hypoglycemia[5] when the food arrives at the duodenum the pancreas will surprise and secretes a large amount of insulin → (Tachycardia, Flushing, Sweating, Colicky pain, hotness and diarrhea may lead to fainting)→ takes 15 mins.
   - Advise the patient to eat less sugar or give him acarbose

4. Alkaline gastritis

[3] Best way for diagnosis unless the pt has a **perforation**. So if a pt - known to have ulcer - came with severe epigastric pain → x-ray to make sure there's no perforation.
[4] To take off G cells → decrease gastrin release.
[5] happens due to huge insulin release.

5. Anemia (MCV : normal 80-100 fl)

- Iron deficiency:
  - MCV < 80 fl (microcytic) and low MCH (hypochromic)
  - Due to decrease in acid production > decrease iron absorption.
- B12 deficiency "megaloblastic or pernicious anemia":
  - MCV > 100 fl (macrocytic) and hypochromic
  - Due to loss of IF production from parietal cell in the fundus.

6. Postvagotomy diarrhea
7. Chronic gastroparesis
8. Pyloric obstruction/stenosis

Ulcer Complications:(Perforation,obstruction & Upper GI bleeding)

	Perforation	Obstruction
SYMPTOMS	- Acute onset of severe unremitting epigastric pain - Sudden, **Severe,** diffuse abdominal pain - Presents as ACUTE ABDOMEN CLINICAL SIGNS (RIGIDITY not GUARDING).	In stomach & duodenal obstruction: - Dull epigastric pain & projectile vomiting of large volumes of undigested food matter - **Vomiting**, +/- weight loss, nonbile stained vomiting.- No abdominal distension, gastric splash[6]
LOCATION	- Occurs in acute ulcers (duodenal mostly) - Gastric perforation is less common and has a strong association with NSAID use. - On the **anterior wall** of the duodenum (duodenal ulcer) or stomach - Anterior ulcers cause perforation, whereas posterior ulcers cause bleeding (due to the Gastroduodenal artery that lies behind the 1st part of the duodenum).	Could be due to stricture formation **3 maneuvers to know the site of obstruction:** - all obstruction present with vomiting - Obstipation\constipation - color of vomit (clear of bile = stomach or duodenum) (dark = small bowel or colon). *if the first presentation is constipation or obstipation then distention &coliky pain, then vomiting → suggest colon obstruction (cancer). *if it starts as distention & coliky pain THEN vomiting → suggest small bowel obstruction (commonest in jejunum) *starts with vomiting **ONLY** → stomach or duodenum
DAIGNOSIS	- **Erect abdominal X-ray**: will demonstrate free air under the diaphragm (85%) [which means air in the peritoneum indicating that there is perforation of the viscus] and fill 400 cc of air by the Nasogastric tube (NGT) (NEVER do EGD) - In comatose either elevate the bed or lateral (right up) because if left up and there is gas could stomach bubble - If you didn't find & fill 400 cc air by NGT then do an X-ray.	- History: (smoking…) - abdominal X-ray you'll; see double bubble - EGD to locate area of obstruction - contrast swallow - Biopsy to rule out cancer
Rx	**- Initial management:** ABC , then, NPO (nothing permouth), IV Fluid, **NGT**(nasogastric tube), ABS (antibiotics) - **Definitive one** is surgical repair (Graham patch[7])	**- Medical treatment** (must make sure pt is taking their medication even if the pain stops) **- Surgical treatment:** 1. Resection (Remove) and anastomose 2. Bypass

- Scenario: patient complaint: first I had constipation, and abdominal distention then I started vomiting. What the diagnosis ?
  A: large bowel obstruction (most likely it's due to cancer)
- patient complaining of coliky pain -/+ constipation → usually small bowel obstruction

[6] If gastric outlet obstruction is clinically suspected, the patient's abdomen may be shaken from side to side in an attempt to elicit a 'succession splash" تسدح المريض وترجه بينما انت حاط السماعه وسط بطنه فإذا فيه ابستروكشن بتسمع صوت زي المويه لما تتزج داخل قروره video

[7] Piece of omentum incorporated into the suture closure of perforation.

Upper gastrointestinal hemorrhage

- The commonest cause of upper GI bleeding is peptic ulcer disease

The differential diagnosis of upper gastrointestinal bleeding:	
Common causes 95%	**Uncommon causes 5%**

• Peptic ulcer 45%
  ○ Duodenal ulcer 25%
  ○ Gastric ulcer 20%
• Esophageal varices 20%
• Gastritis 20%
• Mallory-Weiss syndrome 10%
	• Gastric carcinoma
	• Esophagitis
	• Pancreatitis
	• Hemobilia
	• Duodenal diverticulum

- **Upper gastrointestinal bleeding presents with**:
  - ❖ haematemesis (vomiting fresh blood) /or
  - ❖ Coffee-ground vomitus(is due to vomiting of blood that has been in the stomach long enough for gastric acid to convert hemoglobin to methemoglobin,it's less severe than hematemesis)
  - ❖ melaena (the passage of black tarry stool that has a very characteristic smell).
  - ❖ While hematochezia (the passage of bright-red blood from the rectum) always indicates lower GI bleeding except if there's huge massive peptic ulcer.

- **Management:**

  1- Resuscitation (ABC) "always start with ABC" رجاء رجاء بالاختبار اول شي تقولونه اي بي سي حتى لو المريض ستيبل حتى لو بس تفل دم سووا اي بي سي

  2- Detection and endoscopic treatment (If the cause is an ulcer we can put a clip on it, burn it, use a rubber band or injection of a sclerosing agent to form a clot and stop the bleeding)

  3- Surgical management(Less than 10% of patients bleeding from a peptic ulcer require emergency surgery)

Bleeding site in duodenal ulcers:

- Perforation occurs usually in the anterior walls ulcer.
- Bleeding more commonly occurs in the posterior ulcer, due to the Gastroduodenal artery that lies behind the 1st part of the duodenum, so when bleeding (hematemesis) is seen, we suspect the ulcer to be in the posterior wall of the 1st part of the duodenum.

Other diseases on stomach or duodenum

Zollinger-ellison syndrome (Gastrinoma)	
What is it:	Zollinger-Ellison syndrome is manifested by gastric acid hypersecretion caused by a gastrin producing tumor (gastrinoma). The normal pancreas does not contain appreciable amounts of gastrin. Most gastrinomas occur in the submucosa of the **duodenum**; others are found in the **pancreas** - Gastric hypersecretion + very high no. of ulcers + gastrinoma[8].
Types:	**Malignant:**If it exists alone "gastrinoma only" is usually **malignant.**
	Benign: If it exists in association with multiple endocrine neoplasms type 1(MEN1) (MEN 1) is characterized by a family history of endocrinopathy and the presence of tumors in other glands, especially the parathyroid glands and pituitary. Patients with MEN 1 usually have multiple gastrinomas.
Signs & symptoms	- Symptoms associated with gastrinoma are principally a result of acid hypersecretion → Peptic ulcer disease (often severe) in 95% with Epigastric tenderness , Not recover by medication and , if you Do EGD, you will find a **massive diffuse** ulceration) - Ulcer symptoms are often refractory to large doses of antacids or standard doses of H2 blocking agents.Hemorrhage,perforation, and obstruction are common complications - Some patients with gastrinoma have severe diarrhea from the large amounts of acid entering the duodenum,which can destroy pancreatic lipase and produce steatorrhea
Dx:	**Laboratory finding:** - Elevated serum gastrin MORE THAN 500 pg/ml (G cell in antrum secrete gastrin) **Image study:** - CT or MR scan often demonstrates the pancreatic tumors."If you find GASTRIN LEVEL MORE THAN 500 pg/ml do CT" - Somatostatin-receptor scintigraphy is extremely sensitive for detection of gastrinoma primary and metastatic sites(bc gastrinoma have somatostatin receptor) - EGD:you will find a **massive diffuse** ulceration - Contrast swallow - Portal vein blood sample:Transhepatic portal vein blood sampling to find gradients of gastrin production has been supplanted by the intra-arterial secretin test. Infusion of secretin into the artery supplying a functional gastrinoma causes an increase in hepatic vein gastrin levels. This invasive test is usually reserved for difficult situations.not important at your level
Treatment:	Treatment is mainly surgical !! But you could start with medical tx by giving PPIs, .. until you reach a diagnosis - **Medical Treatment (initial Rx):** Acid control (massive dose of PPI) - **the ideal & appropriate treatment is Surgery:** Distal hemi-gastrectomy and ulcer excision - Find it and get it out , but If you can't find it (50%). Do gastrectomy

[8] G cells tumor.

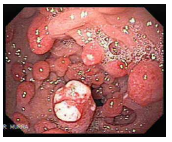
Mallory-weiss syndrome	
What is it:	The lesion consists of a 1- to 4-cm longitudinal tear in the gastric mucosa near the esophagogastric junction; **it usually follows a bout of forceful retching.** - Tear can be in esophageal-gastric junction(EGJ) (most common site), lower esophagus, Cardia and proximal stomach
presentation:	Mallory-Weiss syndrome is responsible for about 10% of cases of acute upper gastrointestinal hemorrhage. Usually caused by **severe retching**, coughing, or **forceful vomiting** (mcq!! Young adult had hematemesis ,with hx of retching → always think about mallory weiss) - Typically, the patient first vomits food and gastric contents,This is followed by forceful retching and then bloody vomitus.
Treatment:	● First Manage by ABC then brief history ● 90% bleeding **stop spontaneously** by ice-water gastric lavage (cold gastric wash[9]). - If it doesn't stop, we perform EGD "to investigate and treat" - If the tear is small, we can burn it (cautery). If not, it will need surgical intervention to repair the tear. ● How control bleeding by scope (cauterize it, band it, clip it, inject or embolize) ○ Never Cauterize in varices

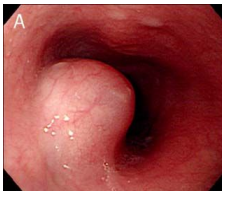
Recall:

What is mallory - weiss syndrome?
Post-retching, post emesis longitudinal tear (submucosa and mucosa) of the stomach near the GE junction; approximately three fourths are in the stomach
What are the causes of a tear?
Increased gastric pressure, often aggravated by hiatal hernia
What are the risk factors?
Retching, alcoholism (50%), 50% of patients have hiatal hernia
What are the symptoms?
Epigastric pain, thoracic substernal pain, emesis, hematemesis
What percentage of patients will have hematemesis?
85%
How is the diagnosis made?
EGD
What is the "classic" history?
Alcoholic patient after binge drinking— First, vomit food and gastric contents, followed by forceful retching and bloody vomitus
What is the treatment?
Room temperature water lavage (90% of patients stop bleeding), electrocautery, arterial embolization, or surgery for refractory bleeding
When is surgery indicated?
When medical/ endoscopic treatment fails (>6 u PRBCs infused)
Can the Sengstaken Blakemore tamponade balloon be used for treatment of Mallory-Weiss tear bleeding?
No, it makes bleeding worse Use the balloon only for bleeding from esophageal varices

[9] To induce vasospasm to stop the bleeding.

Stress gastroduodenitis, stress, stress ulcer & acute hemorrhagic gastritis:	
Stress ulcer:	ulcer due to shock or sepsis
Curling's ulcer:	ulcer due to burns acute gastric erosion results as a complication of severe burns, reduction in plasma volume leads to ischemia and cell necrosis (sloughing) of the gastric mucosa.
Cushing' ulcer:	due to the presence of a CNS tumor or injury (more to perforate, high acid production)
Acute Hemorrhagic Gastritis	This disorder may share some causative factors with the above conditions, but the natural history is different and the response to treatment considerably better. Most of these patients can be controlled medically.

Gastric Polyps	
What is it:	Gastric polyps are single or multiple benign tumors that occur predominantly in the elderly. Those located in the distal stomach are more apt to cause symptoms. 
Types:	- Hyperplastic - treat with Omeprazole - Adenomatous (premalignant) - most serious - Inflammatory - Hamartomatous All are benign except ADENOMATOS is premalignant
presentation:	mainly incidental finding. Rarely, Anemia (Anemia may develop from chronic blood loss or deficient iron absorption)
Dx:	Perform EGD to Rule out malignancy (Whenever gastric polyps are discovered, gastric cancer must be ruled out)
Treatment:	You have to resect the adenomatous type due to its malignant potential. Others are not harmful.

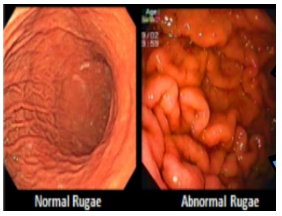
Gastric leiomyomas	
What is it:	Benign smooth muscle tumor Leiomyomas are common submucosal growths that are usually asymptomatic but may cause intestinal bleeding. 
presentation:	90% asymptomatic, less than 1% present with massive bleeding
Dx:	- EGD and CT scan (bulging mass in the mucosa on endoscopy) - Never take biopsy (the capsule will break)
Management:	- by ABC "in case of bleeding" - Surgical wide excision

Diverticula

- **diverticula:** any organ that has lumen is covered by "from in to out side (mucosa-muscle–serosa)". Bulging out or swelling from all the layers (called true diverticula) if mucosa only (it's called false diverticula)
- stomach and duodenum diverticula most likely true diverticula .

Gastric diverticula:	Duodenal diverticula:
• Uncommon • Asymptomatic • Weight loss, diarrhea • It may cause anemia • **Diagnosis:** EGD, X-Ray • Rx:Surgery؟؟؟	• 20% OF POPULATION • Asymptomatic - incidental finding • 90% in the medial aspect of the duodenum • Rare before 40 years of age • Most are solitary and 2.5 cm peri-ampullary of vater • It can cause obstruction, bleeding and inflammation • If it's asymptomatic, we leave it. If there is superficial cancer, we excise it.

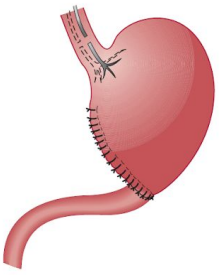
Menetrier's disease

What is it:	acquired, premalignant disease of the stomach characterized by massive gastric folds, excessive mucous production with resultant protein loss Giant hypertrophy of the gastric rugae[10]. Leads to abnormally large secretion of protein-rich mucus and acid 
presentation:	Initially MALNUTRITION only يجيك المريض مايشتكي الا من مالنيوترشن ,اممم عاد حنا دايما رابطين المالنيوترشن بالامعاء فتقوم تفحص الامعاء وماتلقى شي فيها, هنا تقول اممم اكيد انها مينترير ديزيز خل اشيك على المعده ,تشيك عليها بالسكوب تلقى فيها انتشاءات كثيييييره Present with hypoproteinemia, Edema, diarrhea, weight loss
Treatment:	- Atropine (to reduce the secretion) - Omeprazole - H.pylori eradication - Rarely, gastrectomy

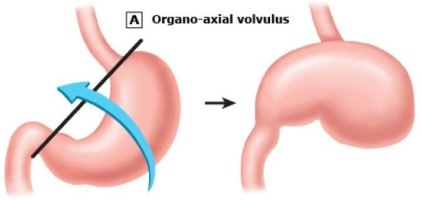
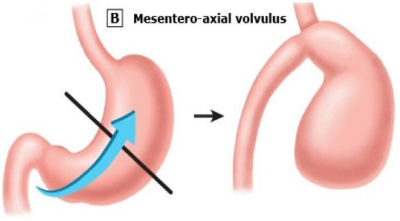
Prolapse of the gastric mucosa

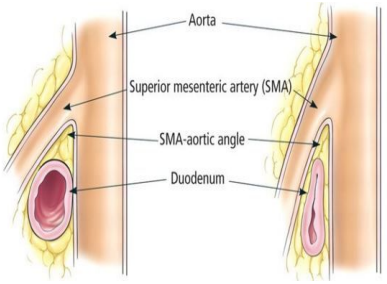
What is it:	When the mucosa of antrum prolapse to duodenum Occasionally accompanies small gastric ulcer
presentation:	Asymptomatic in some pt. In other pts, it could obstructs ampulla of vater causing → obstructive jaundice and Vomiting and abdominal pain
Dx:	**X-ray** : antral folds into duodenum (double ring on X-ray) [not well defined]
Treatment:	Antrectomy with Billroth 1(if not cause symptoms leave it)

Antrectomy and vagotomy (Billroth I)

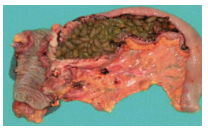


[10] Stomach folds (it's 4 mm in width normally, but in menetrier's disease it's larger)

Gastric volvulus "emergency"	
What is it:	defined as an abnormal rotation of the stomach of more than 180°, which creates a closed-loop obstruction that can results in ischemia Benign disease, but can be lethal
types:	**organo-axial volvulus (longitudinal axis):**[11] - Stomach is closed on two sides (more dangerous) When you introduce NGT it won't pass - More common - Associated with HH (hiatal hernia) 
	mesenteroaxial volvulus (Transverse):(زي الرقاصه;) - Line drawn from the mid lesser curvature to the mid greater curvature - Associated with vomiting (obstruction) - closed from one side (less dangerous) 
presentation:	Presents with: Severe abdominal (epigastric) pain and Borchardt's triad (Borchardt's triad= Vomiting followed by retching and then inability to vomit + Epigastric distention + Inability to pass a nasogastric tube)المعده مسكره لاهو الي قادر يطرش ولا تقدر تدخل لمعدته تيوب
Treatment	Exploratory laparotomy to untwist, and gastropexy

Superior mesenteric artery syndrome	
What is it:	- Obstruction of **the third portion of the duodenum** which is compressed by superior mesenteric artery (SMA) (anteriorly) and Aorta (posteriorly)[12]. - Normally the angle between SMA and Aorta is 50-60 degree and the distance between the - two vessels where the duodenum passes between them is 10–20 mm.In this syndrome angle is less than 45. - Appears after rapid weight loss following injury (happens due loss of mesenteric fat) - Fat is the only thing that lies between the duodenum and the SMA. So when a person is cachexic and chronically ill, the fat will diminish and this will bring the duodenum and SMA closer to each other, leading to the obstruction. 
presentation:	Proximal bowel obstruction symptoms and signs (vomiting)
Dx:	**CT Scan** to look at angle between Aorta & SMA
Treatment:	Pt is cachexic!!! Feed him first, then if it doesn't work do Bypass surgery Chronic obstruction may require section of the suspensory ligament and mobilization of the duodenum, ora duodenojejunostomy to bypass the obstruction

[11] Total rotation along the same axis → blind organ (most dangerous).
[12] Abdominal aorta give 1st celiac branch then SM branch and go down

Bezoar	
What is it:	scientific name of foreign body in the stomach Retained concretions[13] of indigestible foreign material in the stomach (Concretions formed in the stomach)
Types:	**Trichobezoars**: formed from hair غالبا مع المرضى النفسيين تجيهم حالة يقطعوا شعورهم ويبلعوها :) 
	Phytobezoars: vegetable (Indigestible plant material) 
presentation:	Obstruction
Dx:	EGD, X-RAY
Treatment:	SURGICAL REMOVAL

Benign duodenal tumors	
Brunner's gland adenomas	--------
Carcinoid tumors:	Neuroendocrine tumors of the duodenum are often endocrinologically active, producing gastrin, somatostatin,or serotonin. These are tumours of neuroendocrine origin that vary Enormously in their malignant potential. The majority encountered are benign, but occasionally malignant carcinoids can behave aggressively.
Heterotopic gastric mucosa:	presenting as multiple small mucosal nodules, is an occasional endoscopic finding of no clinical significance.
Villous adenomas:	of duodenum may give rise to intestinal bleeding or may obstruct the papilla of Vater and cause jaundice

Regional enteritis of the stomach & duodenum	
causes:	Food poisoning
presentation:	Abdominal pain and diarrhea
Dx:	Clinical
Treatment:	observation

[13] تحجر

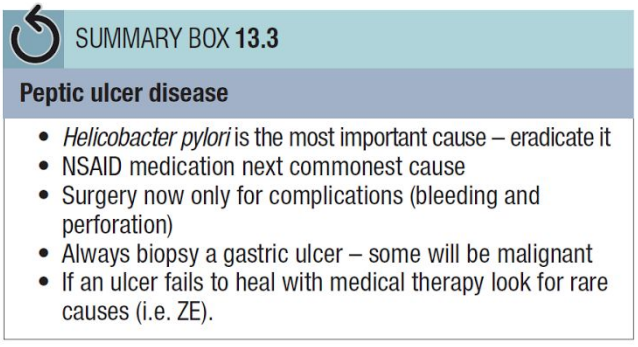
Recall:

What are the possible consequences of PUD?Pain, hemorrhage, perforation,obstruction
Which bacteria are associated with PUD?Helicobacter pylori
What is the treatment?Treat H. pylori with MOC or ACO

- 2-week antibiotic regimens:
  - **MOC**: **M**etronidazole, **O**meprazole,**C**larithromycin (Think: MOCk)
  - Or , **ACO**: **A**mpicillin, **C**larithromycin,**O**meprazole

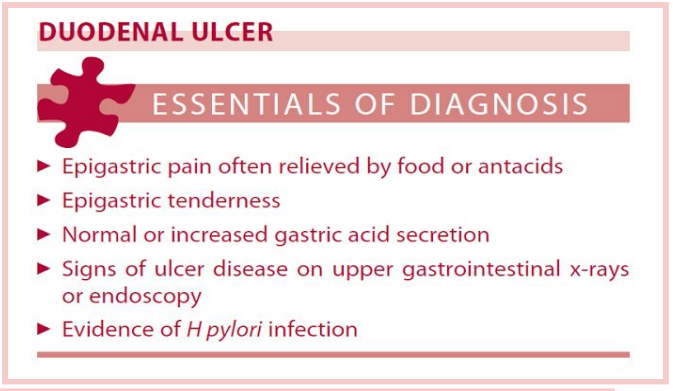
What is the most common location of duodenal ulcer?Most are within 2 cm of the pylorus in the duodenal bulb
What is the classic pain response to food intake?Food classically relieves duodenal ulcer pain (Think: Duodenum Decreased with food)
What is the cause?Increased production of gastric acid
What syndrome must you always think of with a duodenal ulcer?Zollinger-Ellison syndrome
What are the associated risk factors?Male gender, smoking, aspirin and other NSAIDs, uremia, Z-E syndrome,H. pylori, trauma, burn injury
What are the symptoms?Epigastric pain—burning or aching,usually several hours a er a meal (food,milk, or antacids initially relieve pain),Bleeding ,Back pain
What is the differential diagnosis?Acute abdomen, pancreatitis,cholecystitis, all causes of UGI bleeding,Z-E syndrome, gastritis, MI, gastric ulcer,reflux
What artery is involved with bleeding duodenal ulcers?Gastroduodenal artery
Gastric ulcer:
Which is more common overall: gastric or duodenal ulcers?Duodenal ulcers are more than twice as common as gastric ulcers (Think:Duodenal= Double rate)
What is the classic pain response to food?Food classically increases gastric ulcer pain
What is the cause?Decreased cytoprotection or gastric protection (i.e., decreased bicarbonate/mucous production)
Is gastric acid production high or low?Gastric acid production is normal or low!
What are the associated risk factors?Smoking, alcohol, burns, trauma, CNS tumor/trauma, NSAIDs, steroids, shock, severe illness, male gender, advanced age
What is the most common location?~70% are on the lesser curvature; 5% are on the greater curvature
What is the medical treatment? Similar to that of duodenal ulcer—PPIs or H2 blockers, Helicobacter pylori treatment

summaries:


SUMMARY BOX 13.3

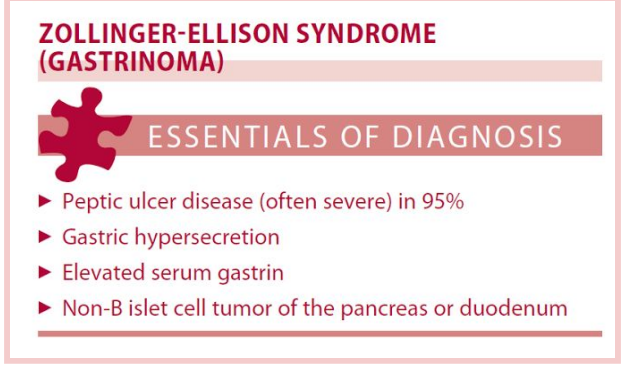
Peptic ulcer disease

- *Helicobacter pylori* is the most important cause – eradicate it
- NSAID medication next commonest cause
- Surgery now only for complications (bleeding and perforation)
- Always biopsy a gastric ulcer – some will be malignant
- If an ulcer fails to heal with medical therapy look for rare causes (i.e. ZE).


DUODENAL ULCER

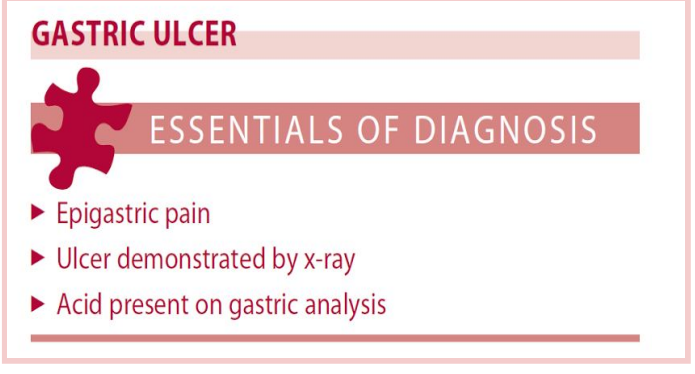
ESSENTIALS OF DIAGNOSIS

- Epigastric pain often relieved by food or antacids
- Epigastric tenderness
- Normal or increased gastric acid secretion
- Signs of ulcer disease on upper gastrointestinal x-rays or endoscopy
- Evidence of *H pylori* infection


ZOLLINGER-ELLISON SYNDROME (GASTRINOMA)

ESSENTIALS OF DIAGNOSIS

- Peptic ulcer disease (often severe) in 95%
- Gastric hypersecretion
- Elevated serum gastrin
- Non-B islet cell tumor of the pancreas or duodenum


GASTRIC ULCER

ESSENTIALS OF DIAGNOSIS

- Epigastric pain
- Ulcer demonstrated by x-ray
- Acid present on gastric analysis

MCQs

Taken from Current diagnosis and treatment

1)The blood supply to the stomach:

A.Typically includes direct branches from the celiac
B.axis and superior mesenteric artery Includes predominant supply of the greater curve of the stomach by the left gastric artery
C.Includes the right gastroepiploic artery, which is usually a branch of the splenic artery
D.May include a posterior gastric artery that is typicall a branch of the splenic artery
E.Is anatomically separate from the blood supply to the spleen

2)The four functions of the stomach include all of these except:

A. It mixes the food and controls delivery into the duodenum.
B. It is the site of the initial stage of protein and carbohydrate digestion.
C. It acts as a reservoir for food.
D. It is the site of the assembly of micelles for nutrient absorption.
E. A few substances are absorbed across the gastric mucosa.

3) Reconstruction of gastrointestinal continuity after resection of portions of the stomach:

A. Usually includes a roux-en-Y reconstruction after distal gastrectomy
B. Cannot be done by Billroth I reconstruction after total gastrectomy
C. Includes a gastroduodenostomy for Billroth II reconstruction
D. Has a risk of duodenal stump leak after Billroth I reconstruction
E. May require conversion to a Billroth II reconstruction if a patient develops bile gastritis after a Billroth I approach

4. Vagotomy:

A. Can impair the appropriate relaxation of the pylorus
B. Has several variations, but each denervates the pylorus
C. Has become more widely applied as ulcer therapy
since the introduction of acid-suppressing medications (H2-blockers and proton pump inhibitors)
D. Impairs gallbladder emptying
E. Can include division of both the left (posterior) and right (anterior) vagus nerve trunks

5. Management of gastric outlet obstruction

A. Is typically required for complications due to distal gastric diverticulae or polyps
B. Should include urgent operation in most patients
C. Initially includes gastric decompression and acid suppression
D. Is commonly required in the management of duodenal ulcer disease
E. Is best managed operatively by distal gastrectomy and Billroth I reconstruction

Answer key:

1 (D) | 2 (D) | 3 (B) | 4 (A) | 5 (C)